Effect of high-selenium wheat on visceral organ mass, and intestinal cellularity and vascularity in finishing beef steers

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Effect of high-selenium wheat on visceral organ mass, and intestinal cellularity and vascularity in finishing beef steers[1]

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ABSTRACT: Twelve crossbred steers (351 ± 24 kg initial BW) were used to determine effects of high-Se wheat on visceral tissue mass, intestinal cell growth, and intestinal cellularity and vascularity. Steers were allotted randomly by BW to one of two treatments consisting of 75% concentrate diets that supplied 1) adequate Se concentration (7 to 12 μg·kg·BW^{-1}·d^{-1}) or 2) high-Se concentration (60 to 70 μg·kg·BW^{-1}·d^{-1}). Diets were similar in composition, including 25% grass hay, 25% wheat, 39% corn, 5% desugared molasses, and 6% wheat middlings supplement on a DM basis. In the Se treatment, high-Se wheat (10 ppm Se, DM basis) was replaced with low-Se wheat (0.35 ppm Se, DM basis). Diets were formulated to be similar in CP and energy (14.0% CP, 2.12 Mcal of NE_m/kg, and 1.26 Mcal NE_g/kg of DM) and were offered daily (1500) to individual steers in an electronic feeding system. Diets were fed at 2.38% BW. After 126 d, steers were slaughtered, and individual visceral tissue weights determined. Concentrations of DNA, RNA, and protein of duodenum, ileum, and total small intestine were not affected ($P \geq 0.33$) by treatment. Similarly, RNA:DNA and protein:DNA ratios in duodenum, jejunum, ileum, and whole small intestine were not ($P \geq 0.33$) affected by feeding high-Se wheat. Conversely, jejunal weight was greater ($P < 0.002$) in steers fed high-Se wheat than in controls (916 vs. 1,427 ± 84 g). Jejunal DNA was increased ($P < 0.04$) in steers fed high-Se wheat (2.95 vs. 3.56 ± 0.19 mg/g), suggesting increased cell number. Concentrations of jejunal RNA and protein were not altered by treatment; however, because the jejunal weight increased in high-Se steers, DNA, RNA, and protein contents (grams) were greater than in control steers ($P < 0.05$). Vascularity of jejunal tissue decreased ($P < 0.10$) with high-Se wheat; however, because jejunal mass was greater for the high-Se wheat treatment, total microvascular volume was not affected by treatment. Percentage of jejunal crypt cell proliferation was not affected ($P = 0.48$) by treatment; however, total number of cells proliferating within the jejunum was increased in steers fed high-Se wheat. Data indicate that the lower jejunal vascularity in the diet high in Se (provided from wheat) may have resulted in increased jejunal mass to meet physiological nutrient demand. Therefore, negative effects of Se level used in this study on productive performance of feedlot steers are not expected.

Key Words: Cellular Growth, Intestine, Selenium, Steer, Wheat

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Introduction

Recent work (Clark et al., 1996; Combs and Lu, 2001) indicates that supranutritional levels of Se (two- to four-fold above normal requirements) can decrease the combined incidence of lung, colorectal, and prostate cancers by as much as 50% in humans. Additionally, research that used rodent cancer models has demonstrated that the positive response to supranutritional (2 to 3 ppm) levels of Se may depend on the molecular form of Se (Finley et al., 2000; Wagner et al., 2000; Finley and Davis, 2001).

In typical diets consumed by North Americans, the major sources of dietary Se are wheat and beef (Schubert et al., 1987; Pennington and Young, 1991). Both wheat and beef have highly variable concentrations of Se that depend on a variety of conditions (Taylor et al., 2002; Hintze et al., 2001). Lawler et al. (2004) recently reported that steers fed 60 to 70 μg Se/d provided as high-Se wheat had similar intakes, gains, and efficien-

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Table 1. Composition (% of DM) of low- and high-Se experimental diets fed to steers

	Treatments	
Ingredient	Low Se	High Se
Coarse dry-rolled corn	39.0	39.0
Alfalfa/grass hay	25.0	25.0
Coarse dry-rolled wheat (low Se)	25.0	25.0
Coarse dry-rolled wheat (high Se)	0.0	25.0
Desugared beet molasses	5.0	5.0
Supplement[a]	6.0	6.0
Dietary Se concentration		
ppm	0.39	2.81
μg Se·kg BW^{-1}·d^{-1}	7 to 12	60 to 70

[a]Supplement (as a percentage of total diet) was composed of 0.93% fine dry-rolled corn, 3.40% wheat middlings, 0.75% limestone, 0.55% urea, 0.11% potassium chloride, 0.21% sodium chloride, 0.01% vitamin E (44 IU/kg), 0.01% tylosin tartrate, 0.02% monensin, 0.01% vitamin A (48,400 IU/kg) and D (4,600 IU/kg).

cies compared with controls that were fed 7 to 12 μg Se/d. They also reported that the Se content of harvested product was high enough to consider beef as a possible option for providing supranutritional levels of Se to human diets. Currently, little data are available regarding the influence of supranutritional levels of Se on growth, cellular proliferation, and vascularity of intestinal tissues. Such data would be relevant to both the practical aspect of producing high-Se beef products and to mechanistic considerations of Se impact on rapidly growing tissue. Therefore, we hypothesized that intestinal cellular proliferation and vascularity will be reduced in feedlot steers by providing supranutritional levels of Se from high-Se wheat.

Materials and Methods

Animals and Diets

Twelve crossbred steers (351 ± 24 kg initial BW) received Ralgro implants (36 mg of zeranol; Schering-Plough Animal Health, Union, NJ) before initiation of the trial and were trained to use Calan Gate individual feeders (America Calan, Inc., Northwood, NH) over a 28-d period. During the training period, steers consumed a common diet of 75% concentrate:25% roughage (DM basis; control diet in Table 1) and were fed at 2.38% of BW. Animal care and use protocols were approved by the NDSU Institutional Animal Care and Use Committee. Feeds used in the adaptation period were purchased from regions of adequate Se concentration. Once training was complete, animals were stratified by BW and assigned randomly to one of two treatments: adequate Se (control), or high Se provided through high-Se wheat (Se-wheat). Dietary Se concentrations were 0.39 and 2.81 ppm, and were fed at 7 to 12 and 60 to 70 μ·kg BW^{-1}·d^{-1} for control and high-Se wheat, respectively. Diets were formulated to contain 14.0% CP, 2.12 Mcal NE_m/kg and 1.26 Mcal NE_g/kg of DM (NRC 1996) and were fed once daily at 1500 at 2.38% of BW. Diets included 8.8 ppm tylosin tartrate (Elanco Animal Health, Indianapolis, IN) and 30 ppm monensin (Elanco Animal Health). High-Se wheat (10.26 ppm Se, as determined by hydride generation atomic absorption spectroscopy) was obtained from a producer near Pierre, SD. High-Se wheat was substituted directly for low-Se wheat to formulate the high-Se wheat treatment (Table 1).

Slaughter Procedure

Steers were fed for 126 d and production data are published elsewhere (Lawler et al., 2004). During the final week of the experiment, animals were assigned to slaughter dates based on live weight. Cattle were removed from feed and water for 24 h before slaughter and final unshrunk live weights were collected 24 h before slaughter. Six steers were slaughtered per day on consecutive days. Visceral organs were obtained immediately after evisceration. Empty body weight (BW minus digesta) was determined after removal of visceral organs. Intestinal segments (duodenum, jejunum, and ileum) were obtained as follows. The duodenum was identified as the segment from the pylorus to a point directly adjacent to the entry of the gastrosplenic vein into the mesenteric vein. The jejunum was the segment from the caudal end of the duodenum to the junction of jejunum and ileum. This junction was determined by measuring 15 cm up the mesenteric vein from the convergence of the mesenteric and ileocecal veins and then up the mesenteric arcade to the point of intestinal intersection. From this point, a 150-cm measurement was made caudally down the small intestine, which was identified as the terminal end of the jejunum and the beginning of the ileum. The 150-cm section was immediately removed for vascular perfusion as described below. The ileum measurement was terminated at the ileocecal junction.

Small Intestine Vascularity

Jejunal tissue was used to evaluate small intestine vascularity or amount of blood vessels in proportion to the amount of tissue. The 150-cm segment of jejunum previously identified was immediately transported to the laboratory and kept hydrated with PBS. The jejunal segment was dissected including the mesenteric vasculature that supplies this portion of jejunum. A primary branch of the mesenteric artery was catheterized using polyethylene tubing (PE-160; o.d. = 1.56 mm, i.d. = 1.14 mm; Becton Dickinson, Sparks, MD). Blood in the vascular arcade was then flushed by perfusion with PBS; next, the area was visualized by perfusing with Evans blue dye (0.05% in PBS, wt/vol; Sigma Chemical Co., St. Louis, MO). The perfused area was regulated by tying or clamping off vessels that provided flow to tissue outside the margins of the desired area. Flow was maintained by manual pressure through the localized tissue until effluent appeared at the mesenteric venous drain-

age. The Evans blue dye was then flushed out with PBS, and the tissue was perfusion-fixed with Carnoy's solution (60% ethanol, 30% chloroform, and 10% glacial acetic acid), followed by a flush with PBS. Finally, the tissue was perfused with a vascular casting epoxy resin (Mercox; 0.8 mL catalyst:5 mL diluent [methyl methanylate]:5 mL resin; all from Ladd Industries, Williston, VT). The tissue was then covered with cheesecloth soaked with PBS, and the Mercox was allowed to harden for 75 to 90 min. Once the Mercox was hardened, 5-mm cross sections of the intestine were immersed in Carnoy's solution for 3 h and then transferred to 70% ethanol until they were embedded in paraffin (Reynolds and Redmer, 1992). The vasculature was visualized immunohistochemically in 5-μm tissue sections with anti-smooth muscle cell α-actin and counterstained with Nuclear Fast red (Sigma Chemical Co.; Redmer et al., 2001). Vascularity was calculated as described by Reynolds and Redmer (1992) and a total of 20 micrographs per steer were analyzed. Total microvascular volume (milliliters) was calculated as mass × vascularity, assuming that 1 g of tissue is equivalent to 1 mL of volume (Reynolds and Redmer, 2001).

Small Intestinal Cellularity

Duodenal, jejunal, and ileal samples were taken for protein, DNA, and RNA analysis as previously described (Scheaffer et al., 2003). These samples consisted of a 2-cm-wide cross section each. Duodenal samples were taken 20 cm cranial from the caudal end of the duodenum. Jejunal samples were taken 155 cm cranial from the caudal jejunal end, and the ileal was taken 40 cm cranial from the ileocecal junction. Samples were frozen in liquid nitrogen and stored at –70°C until analyzed for DNA, RNA, and protein concentrations (Reynolds et al., 1990; Reynolds and Redmer, 1992). Tissue was minced, and 0.5 g was homogenized in Tris, sodium, and EDTA buffer (TNE buffer; 0.05 *M* Tris, 2.0 *M* NaCl, 2 m*M* EDTA, pH 7.4) by using a Polytron with a PT-10s probe (Brinkmann, Westbury, NY). Homogenization was done on ice to a consistent dispersal of tissue (approximately four bursts of 10 s each). Tissue homogenates were analyzed for concentrations of DNA and RNA by using the diphenylamine (Johnson et al., 1997) and orcinol procedures (Reynolds et al., 1990). Standards were DNA Type I from calf thymus and RNA Type IV from calf liver (Sigma Chemical). Protein in tissue homogenates was determined with Coomassie brilliant blue G (Bradford, 1976), with bovine serum albumin (Fraction V; Sigma Chemical) as the standard (Johnson et al., 1997). The concentration of DNA was used as an index of hyperplasia and RNA:DNA ratio was used as an index of hypertrophy (Swanson et al., 2000; Scheaffer et al., 2003). Intestinal section DNA, RNA, and protein content was calculated by multiplying DNA, RNA, and protein concentration by fresh tissue weight (Swanson et al., 2000; Scheaffer et al., 2003).

Jejunal Cell Proliferation

Jejunal samples of 5 to 10 g were taken from the same location as those for DNA, RNA, and protein, and were immediately fixed in 10% formalin and placed into paraffin blocks, sectioned at 5 μm, and then mounted on glass slides for analysis of cellular proliferation as previously described (Fricke et al., 1997; Scheaffer et al., 2003). Tissue sections were treated with blocking buffer consisting of PBS and 1.5% (vol/vol) normal horse serum (Vector Laboratories, Burlingame, CA) for 20 min. Sections of fixed tissues were incubated with mouse anti-proliferating cell nuclear antigen monoclonal antibody (Clone PC-10; Roche Diagnostics Corp., Indianapolis, IN) at 1 μg/mL in blocking buffer (Fricke et al., 1997; Scheaffer et al., 2003). Primary antibody was detected by using a biotinylated secondary antibody (horse anti-mouse immunoglobulin G, Vectastain; Vector Laboratories) and Avidin-Biotin Complex system (Vectastain; Vector Laboratories). Tissue sections were counterstained with Nuclear Fast red to visualize unlabeled nuclei. Number of cells proliferating was calculated by dividing total jejunal DNA by 6.6×10^{-12} g and then multiplying by the percentage of cell proliferation (Baserga, 1985; Zheng et al., 1994).

Intestinal Morphology

Morphology of jejunum was determined on the histological sections by computerized image analysis as previously described (Jin, 1994). A total of 20 villi and their associated length, width, and crypt depth were measured for each steer.

Statistical Analyses

Data were analyzed as a completely randomized design with the GLM procedures of SAS (SAS Inst., Inc., Cary, NC). The model included the fixed effect of treatment.

Results and Discussion

High-Se wheat did not alter duodenal, ileal, small intestinal, large intestinal, stomach complex, mesenteric, and liver mass ($P \geq 0.28$; Table 2). Jejunal mass was greater for high-Se wheat compared with control ($P < 0.01$). Expressing visceral organ mass as grams per kilogram of empty body weight, resulted in similar responses. The major effect of high-Se wheat on visceral organ mass occurred in jejunal tissue, which is one of the most metabolically active tissues among those evaluated in this study (Simon et al., 1982). In general, visceral organs are very active metabolically and represent a substantial amount of maintenance energy consumption (Caton et al., 2000). Metabolic activity of an organ is the product of organ size and metabolic activity per unit of tissue. Simon et al. (1982) reported that pancreas and jejunum had higher fractional rates of

Table 2. Effect of low- or high-Se wheat on digestive tract mass of finishing beef steers

Item	Treatment[a] Control	Se-wheat	SE	*P*-value
No. of observations	6	6		
	g			
Duodenum	512	571	91.0	0.96
Jejunum	916	1,427	84.0	0.01
Ileum	2,693	2,544	173.0	0.56
Small intestine	4,122	4,523	173.0	0.56
Large intestine	2,581	2,531	291.0	0.91
Stomach complex[b]	17,758	17,825	1,275.5	0.97
Mesentary	1,136	1,018	72.0	0.28
Liver	6,732	7,195	323.2	0.34
	g/kg EBW[c]			
Duodenum	1.0	1.1	0.1	0.66
Jejunum	1.8	2.7	0.1	0.01
Ileum	5.2	4.8	0.3	0.40
Small intestine	7.9	8.6	0.4	0.17
Large intestine	4.9	4.8	0.5	0.90
Stomach complex[b]	12.9	13.7	0.4	0.20
Mesentary	2.2	2.0	0.1	0.96
Liver	4.9	4.8	0.5	0.90

[a]Treatments were: Control = 7 to 12 μg Se·kg BW^{-1}·d^{-1} achieved with low-Se wheat; Se-wheat = 60 to 70 μg Se·kg BW^{-1}·d^{-1} achieved with high-Se wheat.
[b]Stomach complex includes rumen, reticulum, omasum, and abomasum.
[c]EBW (empty body weight) = BW minus digesta.

Table 3. Effect of low- or high-Se wheat on DNA, RNA, and protein concentrations in small intestinal tissue (fresh-tissue basis)

Item	Treatment[a] Control	Se-wheat	SE	*P*-value
No. of observations	6	6		
Duodenum				
DNA, mg/g	3.65	4.53	0.83	0.47
RNA, mg/g	3.83	4.07	0.37	0.65
Protein, mg/g	40.60	45.37	6.15	0.60
RNA:DNA	1.2	1.0	0.18	0.51
Protein:DNA	12.81	11.51	2.26	0.69
Jejunum				
DNA, mg/g	2.95	3.56	0.19	0.04
RNA, mg/g	3.45	3.63	0.23	0.59
Protein, mg/g	37.85	40.37	4.70	0.71
RNA:DNA	1.19	1.04	0.10	0.33
Protein:DNA	13.08	11.63	1.72	0.56
Ileum				
DNA, mg/g	5.07	4.42	0.66	0.50
RNA, mg/g	3.90	3.37	0.37	0.33
Protein, mg/g	39.24	44.8	4.72	0.42
RNA:DNA	0.84	0.79	0.07	0.68
Protein:DNA	9.11	11.29	2.00	0.46
Small intestine[b]				
DNA, mg/g	4.41	4.14	0.42	0.66
RNA, mg/g	3.78	3.52	0.26	0.50
Protein, mg/g	38.95	43.23	4.26	0.49
RNA:DNA	0.88	0.87	0.05	0.85
Protein:DNA	9.29	10.94	1.30	0.39

[a]Treatments were: Control = 7 to 12 μg Se·kg BW^{-1}·d^{-1} achieved with low-Se wheat; Se-wheat = 60 to 70 μg Se·kg BW^{-1}·d^{-1} achieved with high-Se wheat.
[b]Small intestine concentration of DNA, RNA, and protein were calculated by adding values for duodenum, jejunum, and ileum contents and dividing by the sum of duodenum, jejunum, and ileum mass.

protein synthesis. In this study, the high-Se wheat seemed to affect tissues with higher metabolic activity.

The RNA content was used as an indicator of protein synthetic capacity (Sainz and Bentley, 1997; Nozière et al., 1999), DNA as an index of tissue hyperplasia (increased cell number), and the protein:DNA and RNA:DNA ratios were used as indices of tissue hypertrophy (increased cell size; Baserga, 1985; Reynolds et al., 1990; Jin et al., 1994). There were no treatment effects ($P \geq 0.33$) in duodenal, ileal, and total small intestine DNA, RNA, and protein concentrations and RNA:DNA, and protein:DNA ratios. This indicates that cells were not increasing in number or size per unit of tissue in response to high-Se wheat. For jejunal RNA and protein concentrations, RNA:DNA, and protein:DNA ratios (Table 3), there were no effects ($P \geq 0.33$) caused by treatment. However, jejunal DNA concentration increased ($P < 0.05$) in steers fed diets containing high-Se wheat. Because DNA per nucleus is relatively constant, this observation indicates that the number of jejunal cells increased per unit of jejunal tissue in response to high-Se wheat. The increase in jejunal cells per unit of jejunal tissue can be explained by the rate of cell proliferation. Even though the percentage of cells proliferating did not differ between control and Se-wheat treatments (20.1 vs. 20.2%; Table 5), both the jejunal DNA concentration (Table 3) and contents (Table 4) increased, resulting in a large increase in the number of cells proliferating (92.1 vs. 168.7×10^9 for control vs. Se-wheat; Table 5). Additionally, the rate of cell death, or apoptosis, also could have changed, as we have shown previously for pig jejunum in response to high dietary fiber (Jin et al., 1994), although we did not examine apoptosis in the present study.

There were no differences ($P \geq 0.33$) in DNA, RNA, and protein contents of duodenum, ileum, and total small intestine (Table 4). Contents of DNA, RNA, and protein of the jejunum increased ($P < 0.02$) when steers were fed diets containing high-Se wheat. The higher jejunal RNA, and protein content for the high-Se wheat treatment was a result of increased jejunal mass (Table 2), whereas increased jejunal DNA content in steers consuming high-Se wheat was due to a combination of increased jejunal mass and DNA concentrations (Table 3).

Cell proliferation (percent) in jejunal tissue was not affected ($P = 0.96$) by treatment (Table 5). However, the total number of proliferating jejunal cells was greater ($P = 0.05$) for steers fed diets that contained high-Se wheat than for controls. However, proliferating cell number was greater for the high-Se treatment, which was caused by the greater jejunal mass in steers

Table 4. Effect of low- or high-Se wheat on total amounts of DNA, RNA, and protein in small intestinal tissue

Item	Treatment[a]		SE	*P*-value
	Control	Se-wheat		
No. of observations	6	6		
Duodenum				
DNA, g	1.71	2.43	0.38	0.21
RNA, g	1.94	2.32	0.38	0.50
Protein, g	21.21	24.99	4.25	0.59
Jejunum				
DNA, g	2.71	5.15	0.49	0.01
RNA, g	3.17	5.25	0.50	0.02
Protein, g	35.11	55.49	4.44	0.01
Ileum				
DNA, g	13.74	11.26	2.03	0.41
RNA, g	10.51	8.53	1.12	0.24
Protein, g	105.81	113.17	14.01	0.72
Small intestine total[b]				
DNA, g	18.15	18.83	2.14	0.83
RNA, g	15.62	16.1	1.61	0.84
Protein, g	162.14	193.65	20.18	0.30

[a]Treatments were: Control = 7 to 12 μg Se·kg BW^{-1}·d^{-1} achieved with low-Se wheat; Se-wheat = 60 to 70 μg Se·kg BW^{-1}·d^{-1} achieved with high-Se wheat.
[b]Small intestine concentration of DNA, RNA, and protein were calculated by adding values for duodenum, jejunum, and ileum.

given the high-Se treatment (Table 2). There were no differences ($P \geq 0.48$) in villus width, villus length, or crypt depth between treatment groups. This indicates that high-Se wheat does not alter intestinal morphology. Because crypts are the principal site of cellular proliferation in the intestinal mucosa (Potten, 1977; Baserga, 1985), jejunal morphology data agree with the cell proliferation results. Vascularity (percent) decreased ($P < 0.10$) when the steers were given the high-Se wheat treatment (Table 5); however, when vascularity was expressed as total microvascular volume, no differences were observed. Total microvascular volume observed in this study indicates that even though the jejunal mass was greater for the high-Se wheat treatment, nutrient transport capacity of jejunal tissue was similar. In other words, the same total vascular volume, and thus transport capacity, was available in the control and steers fed high-Se wheat. It seems that the high-Se treatment also may have lowered turnover of jejunal cells, as jejunal mass was increased even though cell proliferation was not. Thus, the higher jejunal mass of the steers in the high-Se wheat treatment more likely was caused by a lower turnover of cells and not to an increase in cell proliferation. Previous work by Jiang et al. (1999) has shown that Se (3 ppm) as high-Se garlic or selenite decreased intratumoral microvessel density of induced mammary carcinoma in rats. They reported vascularity reductions of 34 and 24% for high-Se garlic and selenite diets, respectively, which is similar to the 20% decrease in vascularity reported in this paper. However, in the work of Jiang et al. (1999), Se diets did not affect vascularity of healthy mammary glands, which disagrees with our results showing a reduction of vascularity of jejunal tissue of healthy beef steers fed 75% concentrate diets that contained 2.81 ppm Se from high-Se wheat. More research in this area to evaluate other tissues as well as the expression of various angiogenic or growth factors may improve our understanding of the responses observed in this study.

Implications

High-Se wheat feedlot finishing diets fed to healthy steers to provide 60 to 70 μg·kg BW^{-1}·d^{-1} decreased vascularity of jejunal tissue. Jejunal mass increased with high-Se wheat, and, as a result, total microvascular volume was not affected by treatment. Although cell proliferation as a percentage of total cells was unaffected by treatment, the increased jejunal mass resulted in more cells proliferating and an associated large increase in jejunal DNA content and mass. In contrast to effects on jejunal tissue, high-Se wheat feedlot finishing diets did not show effects on other tissues measured in this study. Data imply that the jejunal tissue of healthy feedlot steers responded metabolically to diets that provided 60 to 70 μg·kg·BW^{-1}·d^{-1} of Se;

Table 5. Effect of low- or high-SE wheat on cellular proliferation, vascularity, and morphology of jejunal tissue

Item	Treatment[a]		SE	*P*-value
	Control	Se-wheat		
Observations	6	6		
Cell proliferation, %	20.1	20.2	0.03	0.96
Number of proliferating cells × 10^9	92.1	168.7	24.3	0.05
Vascularity, %	12.4	9.9	0.9	0.07
Total microvascular volume, mL[b]	114.6	141.3	14.16	0.21
Morphology				
Villus width, μm	126.7	132.0	11.26	0.74
Villus length, μm	607.0	606.8	48.86	0.99
Crypt depth, μm	443.0	485.0	40.82	0.48

[a]Treatments were: Control = 7 to 12 μg Se·kg BW^{-1}·d^{-1} achieved with low-Se wheat; Se-wheat = 60 to 70 μg Se·kg BW^{-1}·d^{-1} achieved with high-Se wheat.
[b]Calculated as mass × vascularity.

however, absorption capacity (as reflected by total microvascular volume) of the tissue was likely unaltered. Therefore, the Se concentration used in the diet in this study is not expected to have an effect on productive performance of feedlot steers.

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